Remarks on

THE AMBLYOPIA AND ETIOLOGY OF STRABISMUS.

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Read at the Annual Meeting
of the
British Medical Association
at Dublin
August 1887

AMBYLOPIA.

A point of some practical importance, as well as of great theoretical interest, is the nature of the amblyopia of the squinting eye. Very different views are held on this point. By some the amblyopia is looked upon as the cause of the squint, by others as the consequence. Those who entertain the latter view consider the defect of vision to result from disuse, hence the name amblyopia ex anopsia.

The amblyopia of the squinting eye, that is the amblyopia in cases where there are no objective appearances to account for it, in which alone there can be any doubt as to the nature of the connection; varies within tolerably wide limits - We may distinguish two main forms - 1. That in which central fixation is retained. 2. That in which it is lost, or in which at any rate

there is found to be no supremacy of the central over the peripheral portions of the retina. In the first form we find two elements, one of which is permanent, and the other capable of disappearing when the squinting eye is for some days used for fixation. That is to say, the amblyopia may disappear to a certain variable extent, but rarely entirely. Thus one frequently sees that when owing to some injury the good eye has its vision temporarily or permanently impaired, the squinting eye not only takes up the fixation, but rapidly improves in vision - although as a rule falling more or less short of full vision.

Moderate degrees of amblyopia of one eye are frequently met with, indeed much more frequently than are cases of strabismus. It is easy to understand how when the circumstances favouring strabismus exist, that condition is much more likely to become manifest if the one eye is amblyopic, and therefore the value of binocular vision lessened.

A manifest squint does indeed often suddenly make its appearance when the vision of the one eye is accidentally lost or much impaired. There seems every reason then to look upon the permanent element in the first form of amblyopia as not only pre-existing but as one of the chief predisposing causes of the strabismus. Are we then to look upon the recoverable element as caused by disuse? In cases where the squint is associated with hypermetropia, the hypermetropia is often found to be to a much greater extent manifest in the squinting eye, while at the same time little or no attempt is often made to accommodate when that eye is all of a sudden forced to fix on occlusion of the other. Very considerable improvement is got in such cases by the use of the full or nearly full correction. This circumstance then in not a few cases accounts for some part of the defective vision of the

squinting eye. But besides this and constituting usually the main, and it may be, the only portion, of the recoverable element in the amblyopia, is what must be looked upon as a kind of awkwardness in the eye in responding in the full manner of which it is capable, all at once to the normal impressions.. This condition is indeed very similar to left-handedness in so far, as the left hand as a rule, though capable of the same feats as the right, is unable from want of practice, to perform them. The habitual suppression, which takes place to a greater or less extent, of the images falling on the squinting eye, cause it to lose the habit of responding to the full extent to their impressions; but a very short practice, when the fixing eye is thrown out of gear, suffices to overcome this. The recognition of the two elements in the amblyopia of the squinting eye, in most cases at any rate, where the power of

central fixation is retained, is in so far of practical importance - that we may thereby see the uselessness of continuing for months, as is sometimes done - to exercise the squinting eye in order to improve its vision.

A true permanent amblyopia from disuse, is denied by some, because they say that cases, in which, for instance cataract has existed for thirty or forty years, have not been found to have suffered any loss of vision, after successful removal of the cataract. There can be little doubt indeed that when central fixation has been once acquired, it is never lost by circumstances which merely interfere with the formation of distinct images on the macula. It is otherwise, however, with cases in which cataract or any other opacity exists at the time that the supremacy of the macula is acquired. When the cause is removed, although a certain amount of vision is

restored, there is no central fixation. This is always the case in dense congenital cataract and often where there has been a long persisting dense corneal opacity following ophthalmia neonatorum. In the first case, after removal of the cataract and in the second, even when all or nearly all, the opacity has cleared away; the power of central fixation is found to be, and always to remain, absent. In cases of monolateral strabismus which begin early in life, that is during the first months, the squinting eye may be regarded as subjected to the same unfavourable conditions, as far as the acquirement of central fixation is concerned. Fixation with that eye is never called for, and therefore never required. The second form of amblyopia viz: - that in which there is no central fixation possible in the squinting eye is in so far an amblyopia from disuse, in that it is occasioned by

disuse at a time when central fixation is usually acquired. Besides the parallel cases of absence of central fixation from early disuse given above, there is one circumstance which argues strongly for the correctness of this view. We find as already said very frequently unilateral amblyopia where there is no squint, but rarely if ever, an amblyopia without central fixation i.e. of course in cases where there are no objective signs to explain the absence of central fixation.

ETIOLOGY.

In most convergent squints which have existed for some time, we may distinguish between a permanent and an accommodative degree of abnormal convergence. That amount which remains when no effort of accommodation is made, is the permanent amount, whilst the whole amount met with in any case during accurate fixation for any

distance requiring accommodation, is what may be called, the accommodative degree of the squint. The permanent element, the amount of which, in so far as it alone calls for operative correction, it is of some practical importance to ascertain, at all events approximately, is at the same time, that which is the most puzzling in its nature. Most who have studied the question adopt one or other of two explanations which have been advanced. They either with Alfred Graefe contend that an actual shortening, due to structural change, takes place in the internus of the squinting eye, or they hold with Schweigger, that there is gradually induced in this muscle an increase in the amount of its tonic contraction whereby it becomes permanently, although only functionally, shortened. Any view entertained by so great an authority as Alfred Graefe must necessarily command attention, yet

those who carefully read his writings on the subject of strabismus, must be struck with the fact, that neither he nor anyone else, has yet furnished an anatomical demonstration of the supposed structural change in the muscle. Apart from this, and apart also from other objections, which apply as well to Schweigger's explanation, anyone with any experience must have seen lots of cases where a previous existing permanent convergent squint has almost or altogether disappeared. This fact is of itself very strongly suggestive of the incorrectness of the structural change theory, and although it does not of course prove that such a change never takes place, it does most decidedly show that it does not always take place. Schweigger's explanation while it gets over the difficulty of the spontaneous cure of strabismus, is to my mind, equally with the foregoing inconsistent with one circumstance

in connection with squinting. I refer to the disproportion which almost always exists, between the angular deviation of the squinting eye, and the angular insufficiency of its outward movement. I am in the habit in all cases of strabismus of testing the lateral movements of the eyes; and my experience, which I am sure must agree with that of others who have made the same examination, is - that although in perhaps the great majority of cases of convergent strabismus, some restriction in the extent of outward movement is evident in either one or both eyes, the restriction is rarely, if ever, equal to the amount of the squint.

This, I take it, is one of the most important facts which has to be considered in connection with the etiology of strabismus. Another important and more commonly recognised characteristic, that which justifies the name

concomitant, is the practical equality in the degree of deviation of the squint, for all directions of fixation necessitating the same amount of accommodation. Still another is the equality existing between the primary and secondary angles of the squint, that is to say, the degree of the squint when either eye is used for fixation, in all cases where the refraction is the same in the two eyes. These circumstances when considered either singly or combined will be found inconsistent with either the assumption of a weakness of the externus or a spasm of the internus, both of which must necessarily give rise to conditions more allied to what is met with, in paralytic than in ordinary strabismus.

Another explanation which has long been offered and which differs but slightly from Schweigger's, is that the permanent squint is merely the expression for the positim

of equilibrium of the eyes, which instead of being parallelism is convergence. This position, it is held, is assumed when binocular vision for any cause, and the most frequent is of course, amblyopia of the one eye, is not sufficiently useful to assert itself against a disadvantage. According to this view, the spontaneous cure of squint takes place when the anatomical condition of equilibrium becomes altered in such a way as to cause the externi to become relatively more preponderant. It seems very doubtful, however, whether the state of anatomical rest is ever one of convergence, although as V. Graefe long ago pointed out, it is not easy to discover what that position is, in any particular case, as the muscles are continually innervated. From the direction of the orbits and from the divergence met with under chloroform it certainly appears as if the anatomical position of

equilibrium of the eyes was a more or less divergent one.

This position again, which must be looked upon as the position of the starting point for innervation to convergence, no doubt differs, and perhaps not inconsiderably, in different individuals. What gives rise then to the parallelism or approximate parallelism which in most individuals is retained even after one eye is excluded from fixation and there is consequently no desire for binocular vision? If we admit that this position is not necessarily, and from what has been said, not even probably that of anatomical equilibrium, and also that the muscles are constantly innervated, it follows that the position is one rather of what might be called <u>innervation equilibrium.</u> From such a position being constantly required, it becomes eventually firmly established. By constant habit the proper or most useful

disposition of innervation is acquired and permanently and unconsciously maintained.

Donders, having established a connection between convergent strabismus & hypermetropia gave the explanation of this connection in a manner which is familiar to all. He showed what difficulties must necessarily arise on account of the disproportion existing between the accommodative and convergent efforts required for fixation at any particular distance in the case of hypermetropia . There can be little doubt, I think, that this explanation correctly points out, at all events one of the most essential factors in the etiology of strabismus. It has been greatly combated of late and by able opponents, yet on the whole I think, unsuccessfully. No doubt too little stress was originally laid by Donders on the pre-existence of amblyopia in one eye, but that fact does not interfere with the main argument

Another fault as it appears to me, is that the normal, or ideal relation between accommodation and convergence existing in emmetropia is too readily assumed to apply to cases of hypermetropia as well, whereas all who have examined into this connection know, that this is not the case. Instead of the dynamic position, or that found when one eye is occluded being markedly convergent, as it should be even in cases of a moderate degree of hypermetropia, if equal impulses to accommodation and convergence were the rule, the amount of excess of convergence is often found to be slight, or even to give place to divergence. Not unlikely the anatomical position of rest , that is the position of the starting point of convergence, may have something to do with the difference in this respect in different cases. We may assume, however, as more

probable, that the explanation is to be found in differences which exist in the intimacy of the connection between the associated impulses in question; differences which may be partly acquired, and partly inherited. Squinting, we may take it will occur, when the intimacy between the impulses to accommodation and convergence is too close to be with any comfort maintained, without sacrifice of binocular vision. This may be the case even when the vision in both eyes is good, and binocular fusion strong; or as is much more frequently the case, when one eye is amblyopic; but it is by no means always the case, in all cases of hypermetropia, as the disproportion between cases of hypermetropia, and strabismus, of itself shows; although according to the unqualified explanation of Donders, one might suppose that it should be so. But we have still to give an explanation for the

permanent degree of strabismus. It may be formulated as follows: Permanent squinting comes from squinting, just as permanent straight vision comes from looking straight. This which at first sight may appear to flavour of feminine logic, merely expresses the conception that just as parallelism is the equilibrium of innervation in straight eyes so convergence is the position of innervation equilibrium of the squinter. The permanent amount of innervation is in both the result of habit induced by the respective requirements, and in both cases is not unalterable though only liable to slow alteration. This view of the nature of squint, viz: - that it is not due even in its permanent form, to any abnormal state of the muscles, be that a structural or physical change, but merely to an abnormal innervation to convergence and which is really nothing but an extension, so to speak, of Donders theory

to its logical sequences, is that which is held by Hansen-Grut. As a pupil of his I was first taught to look upon convergent strabismus in this way, and have certainly not seen any reason to depart from it. There is not time within the limits of time allotted for this paper, to show how the great majority of cases receive a simple rational explanation on this supposition, and how some cases for which the convergence - innervation theory, as we may call it, is not applicable can be shown to be essentially different from the ordinary cases of concomitant and often wrongly called muscular strabismus convergens. I need only indicate how for instance, it affords an explanation of the complete concomitance of the squint, and of the restriction of movement in the direction opposite to that of the squint. For we know as has been very clearly demonstrated, and explained, by Hering, that an

object in front of, or to either side of, the eyes, and at the same distance from them, necessitates always the same amount of convergence, although in one case, the eye is turned inwards, while in another the same eye is turned outwards. In the first case the inward rotation of the eye is due partly, to a contraction of the internus actuated by a convergent impulse, and partly to one brought about by an impulse to associated movement with the opposite externus. In the second case again while the contraction of the internus as the result of a convergent impulse is still present, it is more than counterbalanced by the externus contraction, which obeys the associated impulse. That is to say, the eyes follow each other to either side, without any alteration in the amount of <u>convergence</u>. In the same way then, in the case of a convergent squint, as the innervation to convergence associated

with a particular distance accommodated for, remains the same, no difference of any amount in the degree of the deviation is met with to either side, that is there is concomitance. Further, the external rotation of the eye must be either greater in range, or affected with greater ease, when unresisted by the contraction of the internus , than when a convergent impulse causes a counteracting contraction of that muscle. We might therefore expect to find some restriction in the outward movement when near objects are fixed, when compared with that possible on the fixation of distant objects. The idea of a permanent convergent innervation as the cause of squint, is certainly then supported by the restriction of the external movement which is often apparent in the non-squinting as well as in the squinting eye.

The conclusions arrived at in this paper may be

shortly stated as follows: - That there is an amblyopia which is, under otherwise favourable conditions the cause of the squint, and also an amblyopia which is the consequence of the squint, though the former is much the more common.

That any explanation of convergent squint which ascribes any abnormality to the muscles of the eye, is inconsistent with the symptoms of squint, and that the permanent degree of a squint is, just as much as the accommodative, due to convergence innervation, the only difference being that the innervation is then permanent.

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